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NEWS 27	Oct 01	CASREACT Enriched with Reactions from 1907 to 1985
NEWS 28	Oct 21	EVENTLINE has been reloaded
NEWS 29	Oct 24	BEILSTEIN adds new search fields
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L1          244 ISCHEMIA (5A) ACTIN

=> s actin (5A)(caudate or cortex)
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L2         1753 ACTIN (5A)(CAUDATE OR CORTEX)

=> s actin (5A) (substantia nigra)
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  53 FILES SEARCHED...
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  69 FILES SEARCHED...
L6       214897 SIZE (4A) (CELL OR NEURON)

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AN   2001:32482  BIOSIS
DN   PREV200100032482
TI   Treatment with nimodipine or mannitol reduces programmed cell
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L4 & ischemia

death and infarct **size** following focal cerebral **ischemia**

AU Korenkov, Alexei I. (1); Pahnke, Jens; Frei, Karl; Warzok, Rolf; Schroeder, Henry W. S.; Frick, Rosemarie; Muljana, Lydia; Piek, Juergen; Yonekawa, Yasuhiro; Gaab, Michael R.

CS (1) Department of Neurosurgery, University of Greifswald, Sauerbruchstrasse, 17487, Greifswald: korenkow@mail.uni-greifswald.de Germany

SO Neurosurgical Review, (September, 2000) Vol. 23, No. 3, pp. 145-150. print.
ISSN: 0344-5607.

DT Article

LA English

SL English

AB The present study was conducted to evaluate the effects of nimodipine and mannitol on infarct size and on the amount of apoptosis after transient focal cerebral ischemia. Focal cerebral ischemia was induced in male Sprague-Dawley rats (weight 300-380 g) by transient occlusion of the right middle cerebral artery (MCAO) using an intraluminal thread model. All animals underwent ischemia for 2 h, followed by 24 h of reperfusion. Group I (n=16) was untreated. Group II (n=16) received 15% mannitol (1 g/kg as bolus) and group III (n=9) received 15 mug/kg/h nimodipine intravenously beginning 15 min prior to MCAO. Twenty-four hours after reperfusion, the brain was taken and sectioned in coronal slices. The slices were stained with H&E and with the transferase dUTP nick-end labeling (TUNEL) technique. Histopathological analysis revealed a significant ($P<0.05$) decrease in infarct size in the striatum with both drugs: mannitol (group II) 25.4+-5.9% and nimodipine (group III) 21.5+-11.0% versus control (group 1) 34.9+-7.0% and in the cortex 2.7+-2.0% (group II) and 6.3+-2.4% (group III) versus control 14.4+-9.0% (group I). The number of apoptotic cells was statistically lower in the therapy groups (group III 9.6, group II 25.8) versus control (group I 57.9) (Mann-Whitney-Wilcoxon U-test $Z>1.96$, $P<0.05$). This study indicates that mannitol and nimodipine provide neuroprotection by preventing both the necrotic and apoptotic components of cell death after transient focal cerebral ischemia and may be effective as neuroprotective drugs for cerebrovascular surgery.

L8 ANSWER 2 OF 8 JICST-EPlus COPYRIGHT 2002 JST

AN 1000629796 JICST-EPlus

TI Melatonin Protects Against **Ischemia**-Reperfusion Induced Astrocytic **Cell** Death and Reduces the **Size** of the Infarction.

AU KUMAZAKI M; YAMAMOTO M; TAKEI N; HIDA H; NISHINO H

CS Nagoya City Univ. Medical School, Aichi, Jpn

SO Jpn J Physiol, (1999) vol. 49, no. Supplement, pp. S235. Journal Code: Z0753A
CODEN: JJPHAM; ISSN: 0021-521X

CY Japan

LA English

STA New

L8 ANSWER 3 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)

AN 1998:883998 SCISEARCH

GA The Genuine Article (R) Number: 131UV

TI Effect of caspase-1 or caspase-3 inhibitor on infarct **size** and apoptotic **cell** death in the **ischemia** reperfused rat heart

AU Okamura T (Reprint); Miura T; Iwamoto H; Shirakawa K; Kawamura S; Ikeda Y; Iwatate M; Matsuzaki M

CS YAMAGUCHI UNIV, UBE, YAMAGUCHI 755, JAPAN

CYA JAPAN

SO CIRCULATION, (27 OCT 1998) Vol. 98, No. 17, Supp. [S], pp. 1353-1353. Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436.

ISSN: 0009-7322.
DT Conference; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 0

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AN 1999:523828 BIOSIS
DN PREV199900523828
TI Effect of caspase-1 or caspase-3 inhibitor on infarct **size** and apoptotic **cell** death in the **ischemia**-reperfused rat heart.
AU Okamura, Takayuki (1); Miura, Toshiro (1); Iwamoto, Hiroshi (1); Shirakawa, Kazuyuki (1); Kawamura, Shuji (1); Ikeda, Yasuhiro; Iwatate, Mitsuo
CS (1) Yamaguchi Univ., Ube Japan
SO Circulation, (Oct. 27, 1998) Vol. 98, No. 17 SUPPL., pp. I260. Meeting Info.: 71st Scientific Sessions of the American Heart Association Dallas, Texas, USA November 8-11, 1998 The American Heart Association . ISSN: 0009-7322.
DT Conference
LA English

L8 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2
AN 1997:481810 CAPLUS
DN 127:170895
TI Selfotel (CGS19755)
AU Schmutz, Markus; Arthur, A.; Faleck, H.; Karlsson, G.; Kotake, A.; Lantwicki, L.; Larue, L.; Markabi, S.; Murphy, D.; Powell, M.; Sauer, D.
CS Research & Development, Pharmaceuticals Division, Ciba, Basel, CH-4002, Switz.
SO Excitatory Amino Acids: Clinical Results with Antagonists (1997), 1-6, 129-152. Editor(s): Herrling, P. L. Publisher: Academic, London, UK. CODEN: 64UIAO
DT Conference; General Review
LA English
AB A review with over 550 refs. Selfotel (CGS19755) is a potent selective and competitive N-methyl-D-aspartate (NMDA) antagonist. Preclinically, selfotel reduced **ischemia**-induced infarct **size** and neuronal **cell** death, antagonized the effects of excitotoxic lesions in the brain, and attenuated neuronal damage following traumatic brain injury at i.p. or i.v. doses ranging from 3 to 40 mg kg-1. In addn. to these neuroprotectant properties, selfotel also exhibited anticonvulsant and anxiolytic activity. Behavioral central nervous system (CNS) effects include ataxia and increased locomotor activity. The compd. was not mutagenic, clastogenic, or teratogenic in rats or rabbits. Similar to other competitive and noncompetitive NMDA antagonists, selfotel produced Olney-type vacuoles in a dose-related manner in rat brain. At present, the clin. significance of these findings in rats is unknown. In humans, the plasma pharmacokinetics of selfotel were linear in the dose range evaluated, and elimination half-life, clearance and vol. of distribution at steady state were independent of dose. There was no appreciable protein binding. Preliminary human data indicate that selfotel rapidly crosses the blood-brain barrier and remains in the cerebral spinal fluid (CSF) for an extended period of time. Doses of up to 3 mg kg-1 i.v. have been evaluated in conscious healthy male subjects, with non-psychotomimetic CNS adverse experiences being the dose-limiting factors. Administration of single doses of up to 2 mg kg-1 of selfotel did not impact the management of neurosurgical patients. However, a no. of these patients also experienced CNS effects. In patients who were conscious following an acute ischemic stroke, doses up to an including 1.5 mg kg-1 i.v. were found safe and were tolerated. Dose-limiting adverse experiences in conscious patients included transient agitation, hallucinations, and confusion at higher doses. In unconscious patients

treated with selfotel following severe closed traumatic brain injury, four bolus doses of up to 5 mg kg-1, administered over a 72 h period, appeared safe and well tolerated. Four international well-controlled clin. trials including approx. 3600 patients, two trials each in acute ischemic stroke and severe traumatic brain injury, are currently underway to evaluate the definitive safety and efficacy of selfotel in improving the functional outcome of patients with these disorders.

L8 ANSWER 6 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:428846 BIOSIS
DN PREV199598443146
TI Recombinant colony stimulating factor-1 reduces infarct **size** and rescues **neurons** in cerebral focal **ischemia**.
AU Berezovskaya, O. (1); Maysinger, D.; Zhai, R. (1); Fedoroff, S.
CS (1) Dep. Pharmacol., Univ. Saskatchewan, Saskatoon, SK S7N 5E5 Canada
SO Society for Neuroscience Abstracts, (1995) Vol. 21, No. 1-3, pp. 540. Meeting Info.: 25th Annual Meeting of the Society for Neuroscience San Diego, California, USA November 11-16, 1995
ISSN: 0190-5295.
DT Conference
LA English

L8 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
AN 1988:73184 CAPLUS
DN 108:73184
TI Dopamine depletion protects striatal neurons from ischemia-induced cell death
AU Clemens, James A.; Phebus, Lee A.
CS Lilly Res. Lab., Lilly Corp. Cent., Indianapolis, IN, 46285, USA
SO Life Sciences (1988), 42(6), 707-13
CODEN: LIFSAK; ISSN: 0024-3205
DT Journal
LA English
AB Infusion of the dopaminergic neurotoxin 6-hydroxydopamine, unilaterally into the substantia nigra of rats, resulted in a unilateral depletion of dopamine in the ipsilateral brain striatum. When these rats were subjected to global forebrain, ischemia, there was a marked protection of the dopamine-depleted striatum from the **ischemia**-induced loss of medium **size neurons** seen in the intact striatum.
These results imply a role for dopamine in the ischemia-induced striatal cell death.

L8 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS
AN 1976:521268 CAPLUS
DN 85:121268
TI Quantitative oxidative enzyme histochemistry of the spinal cord. Part 2. Relation of cell size and enzyme activity to vulnerability to ischaemia
AU Penny, J. E.; Kukums, J. R.; Tyrer, J. H.; Eadie, M. J.
CS Dep. Med., Univ. Queensland, Brisbane, Aust.
SO J. Neurol. Sci. (1975), 26(2), 187-92
CODEN: JNSCAG
DT Journal
LA English
AB Cytophotometric measurements of the activities of 5 enzymes (succinate, malate, and NAD-linked isocitrate dehydrogenases; lactate dehydrogenase, and NADH dehydrogenase) were correlated with cell vol. for neurons in the anterior horn of rabbit lumbar and cervical spinal cord. The data for succinate and isocitrate dehydrogenases indicated that these enzymes were at higher concns. in the smaller neurons, which consist largely of interneurons. No preferential localization to particular sizes of cell could be assigned to the other enzymes studied. The relation between enzyme distribution patterns and their possible role in contributing toward susceptibility to **ischemia** of particular **sizes** of **neurons** is discussed.

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L10  ANSWER 1 OF 6  USPATFULL
AN     2002:164662  USPATFULL
TI     System and method for investigating the effect of chemical and other
       factors on cell movement
IN     Lynes, Michael A., Willington, CT, UNITED STATES
       Knecht, David A., Storrs, CT, UNITED STATES
PI     US 2002086280       A1   20020704
AI     US 2001-2961        A1   20011026 (10)
```

PRAI US 2000-243450P 20001026 (60)
DT Utility
FS APPLICATION
LREP Cummings & Lockwood, P.O. Box 1960, New Haven, CT, 06509-1960
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 1253
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB As disclosed herein, the present invention is directed to a novel system for monitoring cell movement in response to chemotactic and chemokinetic factors. In this system, cells migrate in an under-agarose environment and their position is monitored using a system capable of measuring changes in impedance and other electrical parameters of the system at a target electrode lithographed onto a substrate as the cells arrive at the target. With the disclosed system, the time of arrival of cells at the target electrode is proportional to the dose of the chemoattractant species used to stimulate the cells and can be assessed by changes in resistance at the electrode. The system is readily able to distinguish between wild-type cells and mutants that are deficient in their chemotactic response. In addition, agents that interfere with chemotactic motility can be shown to lead to delayed arrival of cells at the target electrode. The multi-well configuration of the disclosed assay system allows for simultaneous automated screening of many samples for chemotactic or anti-chemotactic activity.

=> d l10 1-6 bib ab

L10 ANSWER 1 OF 6 USPATFULL
AN 2002:164662 USPATFULL
TI System and method for investigating the effect of chemical and other factors on cell movement
IN Lynes, Michael A., Willington, CT, UNITED STATES
Knecht, David A., Storrs, CT, UNITED STATES
PI US 2002086280 A1 20020704
AI US 2001-2961 A1 20011026 (10)
PRAI US 2000-243450P 20001026 (60)
DT Utility
FS APPLICATION
LREP Cummings & Lockwood, P.O. Box 1960, New Haven, CT, 06509-1960
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 1253
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB As disclosed herein, the present invention is directed to a novel system for monitoring cell movement in response to chemotactic and chemokinetic factors. In this system, cells migrate in an under-agarose environment and their position is monitored using a system capable of measuring changes in impedance and other electrical parameters of the system at a target electrode lithographed onto a substrate as the cells arrive at the target. With the disclosed system, the time of arrival of cells at the target electrode is proportional to the dose of the chemoattractant species used to stimulate the cells and can be assessed by changes in resistance at the electrode. The system is readily able to distinguish between wild-type cells and mutants that are deficient in their chemotactic response. In addition, agents that interfere with chemotactic motility can be shown to lead to delayed arrival of cells at the target electrode. The multi-well configuration of the disclosed assay system allows for simultaneous automated screening of many samples for chemotactic or anti-chemotactic activity.

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AN 2000:99857 BIOSIS
DN PREV200000099857
TI The use of quantitative RT-PCR to measure mRNA expression in a rat model of focal **ischemia**: Caspase-3 as a case study.
AU Harrison, David C. (1); Medhurst, Andrew D.; Bond, Brian C.; Campbell, Colin A.; Davis, Robert P.; Philpott, Karen L.
CS (1) Molecular Neurobiology Research, SmithKline Beecham Pharmaceuticals, Third Avenue, New Frontiers Science Park, Harlow, Essex, CM19 5AW UK
SO Molecular Brain Research, (Jan. 10, 2000) Vol. 75, No. 1, pp. 143-149. ISSN: 0169-328X.
DT Article
LA English
SL English
AB Quantitative reverse transcription and polymerisation chain reaction (RT-PCR) using TaqmanTM fluorogenic probes has been used to measure changes in gene expression in the cerebral cortex of rats in the permanent middle cerebral artery occlusion (pMCAO) model of focal **ischemia** . The mRNA levels of three housekeeping genes have been analysed in this model to determine which gene showed least change following experimental insult. In the lesioned **cortex**, beta-**actin** mRNA increased at 24 h, while the levels of cyclophilin and glyceraldehyde-3-phosphate dehydrogenase (GAPDH) did not change. We have also used this methodology to examine modulations in the level of caspase-3 mRNA during focal **ischemia** in the rat. Caspase-3 mRNA showed a 41% increase at 6 h post-MCAO, which was specific to the lesioned cortex. This change became more pronounced with time, showing an increase of 220% at 24 h. This methodology enables changes in mRNA expression to be analysed more sensitively and quantitatively than other available techniques and highlights the need for careful choice of control or housekeeping genes used for RNA comparisons.

L10 ANSWER 3 OF 6 USPATFULL
AN 1998:150936 USPATFULL
TI Uses of estrogen compounds for the treatment of disease
IN Simpkins, James W., Gainesville, FL, United States
PA University of Florida Research Foundation, Inc., Gainesville, FL, United States (U.S. corporation)
PI US 5843934 19981201
AI US 1996-648857 19960516 (8)
RLI Continuation-in-part of Ser. No. US 1993-149175, filed on 5 Nov 1993, now abandoned 76 Ser. No. US 1994-318042, filed on 4 Oct 1994, now patented, Pat. No. US 5554601
DT Utility
FS Granted
EXNAM Primary Examiner: Weddington, Kevin E.
LREP Bromberg & Sunstein LLP
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 11 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 1469
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method is provided for conferring a cytoprotective effect on a population of cells in a male of female subject which includes providing an estrogen compound having insubstantial sex related activity in a pharmaceutical formulation and administering the formulation in an effective dose to a population of cells to confer cytoprotection. The method further includes administering the estrogen compound so as to confer a cytoprotective effect in a subject so as to retard adverse effects of osteoporosis in a male or female subject.

L10 ANSWER 4 OF 6 USPATFULL
AN 1998:54748 USPATFULL
TI Neuron-specific ICAM-4 promoter
IN Kilgannon, Patrick D., Bothell, WA, United States

Gallatin, W. Michael, Mercer Island, WA, United States
PA ICOS Corporation, Bothell, WA, United States (U.S. corporation)
PI US 5753502 19980519
AI US 1996-656984 19960606 (8)
RLI Continuation-in-part of Ser. No. US 1995-481130, filed on 7 Jun 1995, now patented, Pat. No. US 5702917 which is a continuation-in-part of Ser. No. US 1994-245295, filed on 18 May 1994, now patented, Pat. No. US 5700658 which is a continuation-in-part of Ser. No. US 1993-102852, filed on 5 Aug 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John; Assistant Examiner: Brown, Karen E.
LREP Marshall, O'Toole, Gerstein, Murray & Borun
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2173
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB DNA sequences, derived from the 5' region of the human neuron-specific cellular adhesion molecule ICAM-4, which promote neuron-specific gene transcription are disclosed along with polynucleotides comprising the promoter DNA operatively linked to heterologous gene-encoding polynucleotides, expression vectors comprising the promoter DNA, and host cells transformed or transfected with DNA comprising the promoter sequences.

L10 ANSWER 5 OF 6 USPATFULL
AN 96:82674 USPATFULL
TI Methods for neuroprotection
IN Simpkins, James W., Gainesville, FL, United States
Singh, Meharvan, Gainesville, FL, United States
Bishop, Jean, Jacksonville, FL, United States
PA University of Florida, Gainesville, FL, United States (U.S. corporation)
PI US 5554601 19960910
AI US 1994-318042 19941004 (8)
RLI Continuation-in-part of Ser. No. US 1993-149175, filed on 5 Nov 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Weddington, Kevin E.
LREP Bromberg & Sunstein
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN 11 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 1532
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method is provided for conferring neuroprotection on a population of cells using estrogen compounds that have insubstantial sex activity and furthermore, a method is provided that utilizes estrogen compounds in the absence of testosterone for treating neurodegenerative diseases including Alzheimer's disease so as to retard the adverse effects of these disorders, Examples of estrogen compounds that have insubstantial sex activity includes alpha isomers of estrogen compounds such as 17.alpha. estradiol.

L10 ANSWER 6 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 95:22332 SCISEARCH
GA The Genuine Article (R) Number: PY085
TI TUBULIN AND ACTIN MESSENGER-RNAS IN THE YOUNG-ADULT AND THE AGED RAT-BRAIN - EFFECTS OF REPEATED ADMINISTRATION WITH BIFEMELANE HYDROCHLORIDE
AU NISHIBAYASHI S; OGAWA N (Reprint); ASANUMA M; KONDO Y; NORI A
CS OKAYAMA UNIV, SCH MED, INST MOLEC & CELLULAR MED, DEPT NEUROSCI, 2-5-1 SHIKATACHO, OKAYAMA 700, JAPAN (Reprint); OKAYAMA UNIV, SCH MED, INST MOLEC & CELLULAR MED, DEPT NEUROSCI, OKAYAMA 700, JAPAN; OKAYAMA UNIV, SCH

MED, DEPT INTERNAL MED 3, OKAYAMA 700, JAPAN
CYA JAPAN
SO ARCHIVES OF GERONTOLOGY AND GERIATRICS, (NOV/DEC 1994) Vol. 19, No. 3, pp. 265-272.
ISSN: 0167-4943.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 31
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB In an attempt to identify the age-dependent changes in the potential synthesis of cytoskeletal proteins, we investigated changes in messenger RNA (mRNA) of alpha-tubulin and beta-actin in the young-adult and the aged rat brain using Northern blot analysis. alpha-Tubulin mRNA levels in the frontal **cortex** and hippocampus, and beta-**actin** mRNA levels in the hippocampus were significantly decreased in the aged rat brain. Age-dependent decreases in these mRNAs may be related to the neuronal dysfunction associated with aging, in addition to the reduction of several kinds of receptors previously reported. Repeated administration of bifemelane hydrochloride (4-(2-benzylphenoxy)-N-methylbutylamine hydrochloride) for 14 days increased the levels of beta-**actin** mRNA in the frontal **cortex** and the striatum of both young-adult and aged rats,although the effect of bifemelane treatment was smaller and not significant in the aged group, These results suggest that bifemelane treatment may enhance the synthesis of cytoskeletal protein and promote neural plasticity by inducing neurite growth or synapse formation.

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